의과대학 임상의과학과 석사 학위논문

중증 외상에서 병원 사망과 장애에 대한 현장 저산소증과 저혈압의 교호 작용

Interactive Effect between On-Scene Hypoxia and Hypotension on Hospital Mortality and Disability in Severe Trauma

2019 년 2월

서울대학교 대학원
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중증 외상에서 병원 사망과 장애에 대한 현장 저산소증과 저혈압의 교호 작용

지도교수 신 상 도

이 논문을 임상의과학과 석사 학위논문으로

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Interactive Effect between On-Scene Hypoxia and Hypotension on Hospital Mortality and Disability in Severe Trauma

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Background

It is unclear whether effect size of the hypoxia is different on in-hospital mortality and disability according to hypotension status in the field.

Methods

Adult severe trauma (ST) patients during 2012-2013 who were treated by emergency medical services (EMS) and had abnormal revised trauma scores in the field or who had positive trauma triage criteria were analyzed. Exposure was

hypoxia (<94%) measured by EMS. End points were hospital mortality and disability defined as a Glasgow Outcome Scale that decreased by 2 points or more. Multivariable logistic regression with interaction model between hypoxia and hypotension was used for outcomes to calculate the adjusted odds ratios (AOR) with 95% confidence intervals (95%CIs) after adjusting for potential confounders.

Results

A total of 17,406 EMS-ST patients were analyzed. Of those, 2,598 (14.9%) died, and 3,292 (21.5%) were considered disabled at discharge. The total hypoxia group showed higher mortality and disability indices (35.7% and 51.2%) than the non-hypoxia group (10.7% and 15.9%), (each p-value <0.0001). The AOR of hypoxia was 2.15 (1.92-2.40) for mortality and was 1.97 (1.75-2.21) for disability. In the interaction model, AORs for mortality by hypoxia in the hypotensive and non-hypotensive groups were 2.66 (2.32-3.04) and 1.74 (1.61-1.87), respectively (P<0.0001 for interaction). The AORs for disability in the hypotensive and non-hypotensive groups were 2.17 (1.87-2.53) and 1.55 (1.42-1.69), respectively (P<0.0001 for interaction).

Conclusions

The effect of hypoxia was much greater in the hypotensive group than in the non-hypotensive group both in terms of

mortality and disability.

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keywords : Trauma, Hypoxia, Shock, Mortality, Disability

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LEGENDS

TABLE LEGENDS

FIGURE LEGENDS

1. BACKGROUND

Severe trauma is the one of the most important public health issues in developing and developed countries. Worldwide, approximately 5 million people die per year as a result of traumatic injuries.[1] The Global Burden of Disease (GBD) Study 2010 found that injuries caused 11.2% of all disability-adjusted life-years (DALYs) worldwide. [2] Trauma in Korea is the 1st leading cause of death in the population younger than 44 years old according to the Korea National Health Statistics. The total number of death remains at approximately 30,000 per year includes various mechanisms of trauma. The top causes of trauma-related mortalities are traffic accidents and falls according to the annual report on the causes of death statistics. (See the National Statistics Agency Website, http://kostat.go.kr/portal/eng/pressReleases/8/10/index.board. Accessed on Feb. 10, 2017)

In severe trauma, as in other medical conditions, hypoxia at the scene is associated with tissue or end-organ damage because oxygen deficient blood flow can directly injure the tissues. Hypoxia can occur in any situation, such as airway injury, pulmonary contusion due to an external injury to the thorax, insufficient respiration due to altered mental status caused by traumatic brain injury (TBI), asphyxia, drowning, and drug overdose, all of which can interrupt the process of gas exchange in the lungs,.[3-6] Hypoxic conditions (SpO2=<90%) that need supplementary O2 have known to

frequently occur in approximately 38% of the total number of traumatic events.[7]

Hypotension can cause shock, one of the more harmful conditions in trauma, which causes inadequate end-organ perfusion and oxygen delivery.[8] Tissue viability is highly associated with perfusion status. Therefore, hypotension or shock on-scene immediately after a trauma can increase mortality or disability. Approximately 13% of hypotensive trauma patients with systolic blood pressures (SBPs) less than 90 mmHg die within the first 24 hours, with 18% who die at some point during the hospital stay.[9]

Hypoxia and hypotension, individually and combined, have a direct association with mortality or disability in TBI.[10] A previous study showed that a combined status of hypoxia and hypotension on-scene increased mortality more than 4-fold than in the group of patients who were normotensive with normal oxygenation.[11]

Hypoxia with concomitant hypotension can cause a primary ischemic injury and organ failure and can cause a secondary ischemic injury in the other vital organs as well as brain.[12, 13] However, it is uncertain whether hypoxia with hypotension in the field are associated with an increased in in-hospital mortality and disability in patients with multi system trauma. We hypothesized that each risk would be associated with poorer hospital outcomes and that the interaction between hypoxia and hypotension will exacerbate those outcomes. This study aimed to determine the

association between hypoxia and hospital outcomes and to compare the effect size of the hypoxia on the outcomes according to hypotension status.

2. METHODS

The study was approved by the institutional review board (IRB No. 1206-024-412) and by the Korea Centers for Disease Control and Prevention (CDC).

2-1) Study setting

The prehospital trauma care system in Korea is operated and managed by the Central Fire Services (CFS) and the regional fire headquarters of 17 provinces like other emergency medical services. All victims are assessed and managed by the level 1 emergency medical technicians (EMTs) (equivalent to intermediate level of EMT in US) or level 2 EMTs (equivalent to EMT in the US).

Korea has ground EMS units without a tiered response system in cities and a combined ground/helicopter EMS in rural areas. All patients are transported to emergency departments (EDs). The highest provider level is intermediate, which includes intravenous fluid resuscitation for hypotension and endotracheal intubation for traumatic cardiac arrest under online medical control.[14] All basic skills and procedures are under standing orders for level 1 and level 2 EMTs and include the provision of supplementary oxygen via nasal cannula or back-valve mask, spinal immobilization, splinting, and basic wound care. Intermediate skill and procedures are allowed for only level 1 EMTs under direct medical control, including intravenous

fluid infusion for shock status, endotracheal intubation or supraglottic airway insertion for OHCA case due to trauma. Korea's EMS protocol does not allow advanced airway to be performed in TBI without cardiac arrest. IV fluid resuscitation is allowed to be attempted by a Level 1 EMT, but the number of Level 1 EMTs is limited in rural provinces. EMS protocols for the destination hospital were implemented in 2012 using the Korea Prehospital Trauma Triage and Scheme (KPTTS), which has four categories of triage (physiologic, anatomic, mechanism of injury, and clinical) modified from the US CDC' s triage scheme.[15]

The hospital trauma care system has been changed according to the current national plan of the Korean government. All trauma victims are transported to level 1 or level 2 EDs, and level 1 trauma centers, which are covered by emergency physicians or trauma surgeons. High-acuity trauma patients are encouraged to be transported to level 1 or level 2 EDs where definitive care including emergency surgery, embolization, and critical care can be immediately provided. Lower-acuity trauma patients are expected to be transported to level 3 EDs where general physicians can provide appropriate care. Level 1 EDs are staffed with emergency board physicians for 24 hours 7 days. Level 2 EDs are served by emergency physicians including emergency medicine residents for 24 hours 7 days. Level 3 EDs are covered by emergency physicians or general physicians. Level 1 trauma center should have a separated

emergency care unit, operation rooms, and trauma intensive care units in addition to general ED space, and a specialized trauma team. Most trauma centers are established in hospitals with level 1 ED or level 2 ED. All ED levels and trauma center level are designated by the Min. of Health and Welfare of national government and evaluated for the performance and quality annually on the basis of EMS Act. There are designated hospitals with 20 level 1 EDs, 90 level 2 EDs, 300 level 3 EDs, and 6 level 1 trauma centers.[16]

2-2) Study design & data source

This study is a multi-center cross-sectional observational study using the Korean Emergency Medical Services-treated Severe Trauma Registry (EMS-STR) database. The EMS-STR was developed in 2012 through a collaboration between the Korean CFS and the Korean CDC. In 2012, the EMS-STR started in 6 provinces for patients who were transported in 2011 and the expanded to 10 provinces in 2013 for patients who were transported in 2012. There are three basic resources (EMS Patient Care Report (PCR), EMS Trauma Registry, and Hospital Trauma Record Review Registry) available in the EMS-STR. The EMS PCR includes demographic and patient-specific factors, operational variables, time elapse time variables, destination hospital and transport-related information for all injuries. First, the EMS Trauma Registry has more in-depth information on injuriesincluding traumatic and non-traumatic causes

(asphyxia, burn, poisoning, drowning, and other), the injury itself (time/ date, mechanism, activity, and intent), clinical findings (vital signs, mental status, and clinical injury), and prehospital procedures (airway, breathing, circulatory support, spinal immobilization, and wound care) for patients with positive KTPPS criteria assessed by EMTs. Those two data were recorded by EMTs after transporting the patients to the EDs. Third, the Hospital Trauma Record Review Registry has information on clinical findings at the ED (vital signs, mental status, diagnosis codes, and Charlson comorbidity index), hospital procedures (operations and interventions),disposition (discharge, admission, transfer), and outcomes (mortality and disability). All data were collected by professional medical record reviewers from the Korean CDC. There viewers (n=15) are mostly graduated from colleges with a specialty training program on medical record management (3−year or 4−year program). They had worked for data collection from hospital medical records for National Hospital Discharge Survey or National OHCA Survey, which are similar programs in terms of data collection method, the hospital medical records review. The reviewers visited the hospitals where the patients were transported and reviewed the medical records to capture the necessary information.

The data quality management (DQM) committee consisted of epidemiologists, biostatistics experts, emergency medical services physicians, emergency medicine physicians, and

trauma surgeons. All data were registered through a central server at the Korean CDC after being filtered for outliers using a designed protocol. Monthly meetings for medical record reviewers were hosted to maintain the data quality.

2-3) Study population

Patients who were injured by five major mechanisms (traffic accidents, falls, blunt collisions, penetrating injuries, and machinery injuries) were analyzed from January 2012 to December 2013 in 10 provinces in Korea. Patients who were younger than 15 years of age, who had a cardiac arrest before or during prehospital care, and who did not have information on oxygen saturation or systolic blood pressure in the field were excluded.

2-4) Data variables

The exposure variable was hypoxia in the field, which was defined as the SpO2 measured by pulse oximetry by EMTs that was less than 94%, which was defined by the British Thoracic Society.[17] The interaction variable, hypotension, was defined as a systolic blood pressure (SBP) measured by EMTs in the field that was less than 90 mmHg. The EMS trauma protocol recommended that vital signs such as SBP and SpO2 should be measured at least three times; the first, immediately after arrival to the scene, the second, during ambulance transport, and the third, at the time of arrival to the ED. We used the baseline measurements of SpO2 and

SBP on-scene.

Confounding variables included age, sex, Charlson comorbidity index, mechanism of injury, elapsed time interval from event to EMS arrival, mental status in the field, prehospital airway and ventilatory support, and new injury severity score (NISS). Age was categorized as adult (15-64 years) and older adults (65 years or older). The Charlson comorbidity index was categorized into two groups(none vs. one or more) according to the number of co-morbidities.[18, 19] The mechanisms of injury included traffic accidents, falls, collisions, penetrations, and machinery accidents. Mental status was categorized with AVPU scale four groups; alert (A), response to verbal (V), response to pain (P), and unresponsiveness (U). Prehospital airway and ventilatory support were classified into four groups; advanced airway management including supraglottic airwaysand endotracheal intubation, positive pressure bag-valve mask ventilation, oral airway management with supplementary oxygen via a nasal cannula or a non-rebreather mask, or oral airway managements without supplementary oxygen. Injury severity was categorized into four groups; mild (NISS 1-8), moderate (NISS 9-15), severe (NISS 16-24), and critical (NISS 25-75).[20] Hospital care and outcome variables included cardiopulmonary resuscitation in the ED, surgery under general anesthesia, admission to the intensive care unit, mortality, and disability. Disability was measured twice: at the time of the injury and at hospital discharge. Both

times that disability was measured the Glasgow outcome scale (GOS) was used and includes domains of death, vegetative status, severe disability (dependent status), moderate disability (non-dependent status), and good recovery.[21] Two GOSs were retrospectively measured by medical record reviewers on the basis of medical records written by duty physicians, duty surgeon, or duty nurse.

2-5) Outcome measure

The primary end point was hospital mortality and the secondary end point was a worsened disability. Worsened disability was defined when patients died before discharge or had a decrease in their GOS score by 2 points between the GOS score that was recorded at discharge and the GOS score that occurred atthe time of the injury. Current many literatures on trauma showed the proportion of older adults, or potentially disabled population such as patients with stroke, or patients with previous severe trauma was higher in study population. [22, 23] Therefore, some patients might have had disability due to previous trauma or disease. Single measurement of disability at discharge may influenced by previous disability level. To know the exact impact of current trauma on disability, both pre-event status and post event status should be considered. To measure the impact of trauma on disability, we used the "worsened disability" as an outcome in this study which was operationally defined without a previous validation study.

2-6) Statistical analysis

A descriptive analysis was performed to compare the distribution of risk factors between exposure groups using chi-square tests for categorical variables and Wilcoxon rank sum tests for continuous variables. To determine the outcome trend by exposure variables, we used a restricted cubic spline analysis and evaluated the graph.

For the analysis of the association between exposures and outcomes, a multivariable logistic regression was used for outcomes after adjusting for confounders in the final model. Those confounders included age, sex, Charlson comorbidity score, mechanism of injury, response time interval from the call for the ambulance and the arrival to the scene, mental status in the field, prehospital airway management, and NISS groups. The adjusted odds ratios (AOR) with 95% confidence intervals (95%CIs) were calculated to measure the effect of hypoxia and hypotension compared to a normal group. To compare the effect size of the hypoxia according to hypotensive status, an interaction analysis was performed using the interaction term (hypoxia*hypotension) that was added to the final multivariable logistic model.

We performed three types of sensitivity analysis for the different study population. The first sensitivity analysis was performed for patient groups with very low values (SpO2 and SBP) that could not be correctly measured. We excluded patients who had a SpO2 of less than 60% and an SBP of less than 60 mmHg. The second sensitivity analysis was

performed for the study population with a different definition of hypoxia using a cut-off (SpO2<90%). The third sensitivity analysis was performed forthe patients without traumatic brain injury (TBI). TBI patients were defined according to the International Classification of Disease (ICD)-10th version, including all patients with diagnosis codes of S06.0-S06.9 (concussion, traumatic cerebral edema, diffuse brain injury, focal brain injury, epidural hemorrhage, traumatic subdural hemorrhage, traumatic subarachnoid hemorrhage, intracranial injury with prolonged coma, other intracranial injuries, and unspecified intracranial injury).

We used the multivariable logistic regression analysis for the association between hypoxia or hypotension and outcomes adjusting for same confounders in the main analysis. Additionally, the interaction analysis using the same model of main analysis for the different study population was performed to test the interactive association between hypoxia and hypotension for outcomes.

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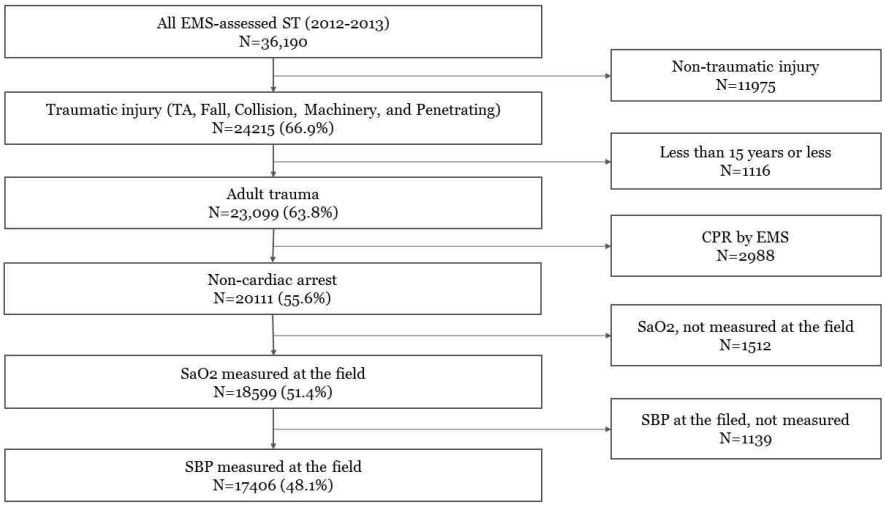
3 . RESULTS

3-1) Demographic findings

A total of 36,190 trauma patients in study period were treated by EMS and of those, 17,406 patients were analyzed, with 11,975 patients who were excluded because of another mechanism of injury. Pediatric patients accounted for 1,116, while 2,988 received CPR by EMS providers, 1,512 lacked a SpO2value, and 1,139 lacked an SBP value. (Figure 1) The hypoxia versus the non-hypoxia group showed a higher mortality (35.7% versus 10.7%) and worsened disability (51.2% vs. 15.9%), respectively (all p values <0.0001). The hypotensive versus the non-hypotensivegroup showed higher mortality (34.2% versus 12.4%) and worsened disability (41.0% vs. 19.0%), respectively (all p values <0.0001). (Table 1)

Of the 17,406 patients, those who were male, older adults, were involved in a traffic accident, had a Charlson comorbidity index of 1 or more, were hypotensive, were in the responsive to pain and in the unresponsive group, had response time intervals greater than 16 min., were in receipt of supplementary oxygen, had CPR that was performed in the ED, had a surgical intervention except musculoskeletal surgeries, were in the ICU care group, and had higher NISS scores all showed a more hypoxic status (all p values <0.0001). (Table 1).

Figure 1. Patient flow



EMS, emergency medical services

ST, severe trauma

TA, traffic accident

SBP, systolic blood pressure

CPR, cardiopulmonary resuscitation

Hypotensive patients were more likely to be involved in traffic accidents or have penetrating trauma, had hypoxia, had either an alert or an unresponsive mental status, had a response time interval greater than 16 min., had advanced airway management or bag-valve mask ventilation, had CPR in the ED, had abdominal surgeries, and had moderate and severe NISS scores (all p values <0.0001). However, gender, age group, and surgeries for the thorax and the musculoskeletal systems did not produce any significant differences. Higher Charslon comorbidity indices, brain

surgeries, ICU care, and critical NISS groups were likely to occur in the non-hypotensive group (all p values <0.0001). (Table 2)

Figure 2 showed the outcome trend according to SpO2 increase which showed the linear association between probability of outcomes (death and worsened disability) and SpO2 value in the adjusted model. There was no clear cut-off value for discrimination between lower SpO2 versus higher SpO2 for outcome. The cubic spline without adjustment showed four knots (82, 95, 97, 98, and 100).

Table 1. Demographic findings between hypoxic and non-hypoxic groups

Variables		All		Hypoxic		Non-hypoxic		p-value
		N	%	N	%	N	%	
All		17406	100.0	2922	100.0	14484	100.0	
Gender								<.0001
	Male	12280	70.6	2205	75.5	10075	69.6	
	Female	5126	29.4	717	24.5	4409	30.4	
Age group, years								<.0001
	Adult (15-64)	12859	73.9	1987	68.0	10872	75.1	
	Elderly (>=65)	4547	26.1	935	32.0	3612	24.9	
	Median (Q1-Q3)	51 (36-64)		54 (42-68)		51 (35-63)		<.0001
Mechanism of Injury								<.0001
	Traffic accident	8394	48.2	1599	54.7	6795	46.9	
	Fall	6515	37.4	1004	34.4	5511	38.0	
	Collision	1362	7.8	148	5.1	1214	8.4	
	Penetration	886	5.1	144	4.9	742	5.1	
	Machinery	249	1.4	27	0.9	222	1.5	
Charlson comorbidity index								0.0095
	None	16266	93.5	2699	92.4	13567	93.7	
	One or more	1140	6.5	223	7.6	917	6.3	
Systolic blood pressure								<.0001
	<90 mmHg	2013	11.6	893	30.6	1120	7.7	
	>=90 mmHg	15393	88.4	2029	69.4	13364	92.3	
Mental status								<.0001
	Alert	7007	40.3	594	20.3	6413	44.3	
	Verbal response	6448	37.0	812	27.8	5636	38.9	
	Pain response	2917	16.8	825	28.2	2092	14.4	
	Unresponsiveness	1034	5.9	691	23.6	343	2.4	
RTI, minutes								<.0001
	0=<RTI<4	1428	8.2	205	7.0	1223	8.4	

	4<=RTI<8	8268	47.5	1277	43.7	6991	48.3	
	8<=RTI<12	4009	23.0	685	23.4	3324	22.9	
	12<=RTI<16	1805	10.4	330	11.3	1475	10.2	
	16<=RTI	1896	10.9	425	14.5	1471	10.2	
	Median (Q1-Q3)	7 (5-11)		7 (5-12)		7 (5-10)		<.0001
Prehospital airway management								<.0001
	Advanced airway	41	0.2	24	0.8	17	0.1	
	BVM with oxygen	372	2.1	285	9.8	87	0.6	
	PV with oxygen	8421	48.4	2061	70.5	6360	43.9	
	PV without oxygen	8572	49.2	552	18.9	8020	55.4	
CPR in ED								<.0001
	No	16682	95.8	2455	84.0	14227	98.2	
	Yes	724	4.2	467	16.0	257	1.8	
Surgery								
	Brain	899	5.2	218	7.5	681	4.7	<.0001
	Chest	133	0.8	57	2.0	76	0.5	<.0001
	Abdomen and pelvis	290	1.7	83	2.8	207	1.4	<.0001
	Musculoskeletal	1639	9.4	280	9.6	1359	9.4	0.7360
Intensive care unit								<.0001
	No	14209	81.6	2097	71.8	12112	83.6	
	Yes	3197	18.4	825	28.2	2372	16.4	
NISS								<.0001
	1<=NISS<9	9609	55.2	954	32.6	8655	59.8	
	9<=NISS<16	2809	16.1	480	16.4	2329	16.1	
	16<=NISS<25	2377	13.7	592	20.3	1785	12.3	
	25<=NISS<=75	2330	13.4	818	28.0	1512	10.4	
	Unknown	281	1.6	78	2.7	203	1.4	
Outcomes								
	Mortality	2598	14.9	1044	35.7	1554	10.7	<.0001
	Disability*	3542	21.5	1270	51.2	11643	15.9	<.0001

RTI, response time interval; BVM, bag-valve mask; PV, passive ventilation via facial mask or nasal prong; CPR, cardiopulmonary resuscitation; ED, emergency department; NISS, new injury severity score

* Missing values were excluded in denominator and numerator (total n=951, 634 for non-hypoxic group and 317 for hypoxic group)

Table 2. Demographic findings between hypotensive and non-hypotensive groups

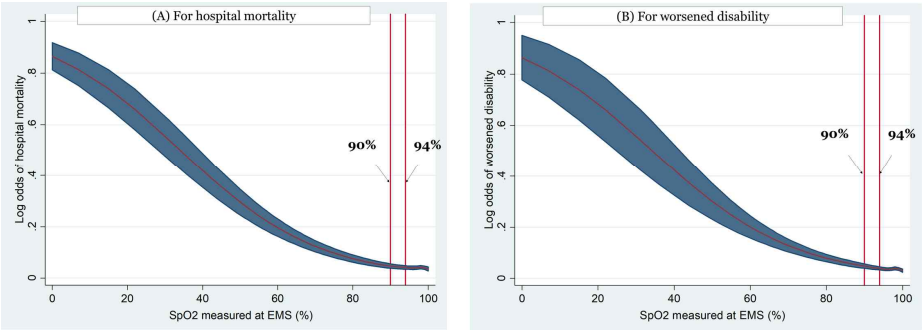
Variables		All		Hypotensive		Non-hypotensive		p-value
		N	%	N	%	N	%	
All		17406	100.0	2013	100.0	15393	100.0	
Gender								0.2749
	Male	12280	70.6	1399	69.5	10881	70.7	
	Female	5126	29.4	614	30.5	4512	29.3	
Age group, years								0.7932
	Adult (15-64)	12859	73.9	1492	74.1	11367	73.8	
	Elderly (>=65)	4547	26.1	521	25.9	4026	26.2	
	Median (Q1-Q3)	51 (36-64)		51 (36-64)		52 (36-64)		0.8472
Mechanism of Injury								<.0001
	Traffic accident	8394	48.2	1000	49.7	7394	48.0	
	Fall	6515	37.4	624	31.0	5891	38.3	
	Collision	1362	7.8	146	7.3	1216	7.9	
	Penetration	886	5.1	209	10.4	677	4.4	
	Machinery	249	1.4	34	1.7	215	1.4	
Charlson comorbidity index								0.0352
	None	16266	93.5	1903	94.5	14363	93.3	
	One or more	1140	6.5	110	5.5	1030	6.7	
Hypoxia								<.0001
	SpO2<94%	2922	16.8	893	44.4	2029	13.2	
	SpO2>=94	14484	83.2	1120	55.6	13364	86.8	
Mental status								<.0001
	Alert	7007	40.3	1080	53.7	5927	38.5	
	Verbal response	6448	37.0	216	10.7	6232	40.5	
	Pain response	2917	16.8	198	9.8	2719	17.7	
	Unresponsiveness	1034	5.9	519	25.8	515	3.3	
RTI, minutes								<.0001
	0=<RTI<4	1428	8.2	128	6.4	1300	8.4	
	4<=RTI<8	8268	47.5	873	43.4	7395	48.0	
	8<=RTI<12	4009	23.0	476	23.6	3533	23.0	
	12<=RTI<16	1805	10.4	218	10.8	1587	10.3	
	16<=RTI	1896	10.9	318	15.8	1578	10.3	
	Median (Q1-Q3)	7 (5-11)		8 (5-12)		7 (5-10)		<.0001
Prehospital airway management								<.0001
	Advanced airway	41	0.2	10	0.5	31	0.2	
	BVM with oxygen	372	2.1	175	8.7	197	1.3	
	PV with oxygen	8421	48.4	945	46.9	7476	48.6	
	PV without oxygen	8572	49.2	883	43.9	7689	50.0	
CPR in ED								<.0001
	No	16682	95.8	1757	87.3	14925	97.0	
	Yes	724	4.2	256	12.7	468	3.0	
Surgery								
	Brain	899	5.2	58	2.9	841	5.5	<.0001
	Chest	133	0.8	21	1.0	112	0.7	0.1333
	Abdomen and pelvis	290	1.7	59	2.9	231	1.5	<.0001
	Musculoskeletal	1639	9.4	200	9.9	1439	9.3	0.3942
Intensive care unit admission								<.0001
	No	14209	81.6	1724	85.6	12485	81.1	
	Yes	3197	18.4	289	14.4	2908	18.9	
NISS								<.0001

	1<=NISS<9	9609	55.2	1019	50.6	8590	55.8	
	9<=NISS<16	2809	16.1	354	17.6	2455	15.9	
	16<=NISS<25	2377	13.7	297	14.8	2080	13.5	
	25<=NISS<=75	2330	13.4	259	12.9	2071	13.5	
	Unknown	281	1.6	84	4.2	197	1.3	
Outcomes								
	Mortality	2598	14.9	689	34.2	1909	12.4	<.0001
	Disability	3542	21.5	781	41.0	2761	19.0	<.0001

RTI, response time interval; BVM, bag-valve mask; PV, passive ventilation via facial mask or nasal prong; CPR, cardiopulmonary resuscitation; ED, emergency department; NISS, new injury severity score

* Missing values were excluded in denominator and numerator (total n=951, 843 for non-hypotensive group and 108 for hypotensive group)

Figure 2. Restricted cubic splines of SpO2 values recorded by EMS and the log odds of in-hospital mortality and worsened disability



The log odds of hospital mortality were adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, prehospital airway management, and new injury severity score.

(A) For in-hospital mortality

(B) For worsened disability

3-2) Main analysis

From the multivariable logistic regression analysis for in-hospital mortality, the AORs (95%CIs) for hypoxia were 2.15 (1.92-2.40). For worsened disability, the AORs (95%CIs) for hypoxia were 1.97 (1.75-2.21). For in-hospital mortality, the AORs (95%CIs) for hypotension were 2.89(2.54-3.29) and for worsened disability, the AORs (95%CIs) for hypotension were 2.15 (1.86-2.49) in the model. (Table 3)

Table 3. Multivariable logistic regression analysis by hypoxia and hypotension for in-hospital mortality and disability

Outcomes	Exposure		Total	Outcome (+)		Adjusted model*		
			N	N	%	AOR	95% CI	
Mortality	Total		17,406	2,598	14.9			
	Hypoxia							
		No	14,484	1,554	10.7	1.00		
		Yes	2,922	1,044	35.7	2.15	1.92	2.40
	Hypotension							
		No	15,393	1,909	12.4	1.00		
		Yes	2,013	689	34.2	2.89	2.54	3.29
Disability	Total		16,455	3,542	21.5			
	Hypoxia							
		No	13,850	2,207	15.9	1.00		
		Yes	2,605	1,335	51.2	1.97	1.75	2.21
	Hypotension							
		No	14,550	2,761	19.0	1.00		
		Yes	1,905	781	41.0	2.15	1.86	2.49

AOR, adjusted odds ratio; 95%CI, 95% confidence interval

* Adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, prehospital airway management, and new injury severity score

3-3) Interaction analysis

The AORs (95%CIs) for hypoxia stratified by whether the patient was non-hypotensive or hypotensive for in-hospital mortality were 1.74 (1.61-1.87) and 2.66 (2.32-3.04),

respectively (p for interaction <0.0001). The AORs (95%CIs) for hypoxia stratified by whether the patient was non-hypotensive or hypotensive for worsened disability were 1.55(1.42-1.69) and 2.17 (1.87-2.53), respectively (p for interaction <0.0001). (Table 4)

Table 4. Interaction analysis of hypoxia and hypotension on mortality and disability using multivariable logistic regression analysis

Outcome	Hypoxia	Hypotension						p value for interaction
		No			Yes			
		AOR	95% CI		AOR	95% CI		
Mortality								<0.0001
	No	1.00						
	Yes	1.74	1.61	1.87	2.66	2.32	3.04	
Disability								<0.0001
	No	1.00						
	Yes	1.55	1.42	1.69	2.17	1.87	2.53	

AOR, adjusted odds ratio; 95% CI, 95% confidence interval
Multivariable logistic regression analysis, adjusted for adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, prehospital airway management, new injury severity score, and interaction term(hypoxia*hypotension)

3-4) Sensitivity analysis for different study population

First, we performed a sensitivity analysis for the overall group of patients (N=17406), excluding patients with SpO2 values of less than 60% (N=463) and SBPs of less than 60 mmHg (N=177). For in-hospital mortality, the AORs (95%CIs) by hypoxia and by hypotension were 1.39 (1.23-1.56) and 1.40 (1.19-1.64), respectively. For worsened disability, the AORs (95%CIs) for hypoxia and for hypotension were 1.52 (1.35-1.72) and 1.37 (1.16-1.61), respectively. Second, we also tested the association between

hypoxia and outcomes of study population using a different definition of hypoxia (SpO2 less than 90%). For hospital mortality, the AORs (95%CIs) by hypoxia with a SpO2 of less than 90% and hypotension were 2.91 (2.55-3.32) and 2.89 (2.54-3.29), respectively. For worsened disability, the AORs (95%CIs) by hypoxia using the same definition and hypotension were 2.97 (2.54-3.47) and 2.15 (1.86-2.49), respectively. Third, we tested the association between hypoxia and hypotension on outcomes for study population without TBI. For hospital mortality, the AORs (95%CIs) by hypoxia and hypotension were 2.57 (2.16-3.06) and 2.89 (2.54-3.29), respectively. For worsened disability, the AORs (95%CIs) by hypoxia and hypotension were 2.28 (1.93-2.69) and 2.15 (1.86-2.49), respectively. (Table 5)

We tested the interactive association between hypoxia and hypotension for outcomes in above different study populations. First, the AORs (95%CIs) by hypoxia according non-hypotensive and hypotensive statuses on in-hospital mortality in study population, excluding patients with SpO2 values of less than 60% and SBPs of less than 60 mmHg, were 1.25 (1.15-1.36) and 1.38 (1.19-1.62), respectively. (p for interaction=0.0148) The AORs (95%CIs) for hypoxia for the effects of non-hypotensive and hypotensive statuses on worsened disability in the same study population were 1.29 (1.18-1.41) and 1.40 (1.19-1.65), respectively. (p for interaction=0.0617) Second, the AORs (95%CIs) by hypoxia according non-hypotensive and hypotensive statuses on

in-hospital mortality in of study population using a different definition of hypoxia (SpO2 less than 90%) were 1.90 (1.74-2.06) and 3.16 (2.75-3.62), respectively. (p for interaction <0.0001) The AORs (95%CIs) for hypoxia for the effects of non-hypotensive and hypotensive statuses on worsened disability in the same study population were 1.93 (1.76-2.12) and 2.81 (2.39-3.29), respectively. (p for interaction <0.0001) Third, the AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on in-hospital mortality for study population without TBI were 1.78 (1.60-1.97) and 2.44 (2.05-2.90), respectively. (p for interaction <0.0001) The AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on worsened disability were 1.65 (1.49-1.83) and 2.09 (1.76-2.49), respectively. (p for interaction <0.0001) (Table 6)

Table 5 Multivariable logistic regression analysis by hypoxia and hypotension for in-hospital mortality and disability for sensitivity analysis

Group	Outcomes	Exposure		Total	Outcome		Adjusted model*		
				N	N	%	AOR	95CI	
Study population excluding patients with SpO2<60% or SBP<60 mmHg									
	Mortality	Total		16766	2146	12.8			
		Hypoxia							
			No	14353	1548	10.8	1.00		
			Yes	2413	598	24.8	1.39	1.23	1.56
		Hypotension							
			No	15366	1903	12.4	1.00		
			Yes	1400	243	17.4	1.40	1.19	1.64
	Disability	Total		15837	3068	19.4			
		Hypoxia							
			No	13722	2197	16.0	1.00		
			Yes	2115	871	41.2	1.52	1.35	1.72
		Hypotension							
			No	14527	2747	18.9	1.00		
			Yes	1310	321	24.5	1.37	1.16	1.61
Study population using a new definition of hypoxia (<90%)									
	Mortality	Total		17406	2598	14.9			
		Hypoxia							
			No	15761	1817	11.5	1.00		
			Yes	1645	781	47.5	2.91	2.55	3.32
		Hypotension							
			No	15393	1909	12.4	1.00		0.00
			Yes	2013	689	34.2	2.89	2.54	3.29
	Disability	Total		16455	3542	21.5			
		Hypoxia							
			No	15006	2589	17.3	1.00		
			Yes	1449	953	65.8	2.97	2.54	3.47
		Hypotension							
			No	14550	2761	19.0	1.00		
			Yes	1905	781	41.0	2.15	1.86	2.49
Study population without patients with traumatic brain injury									
	Mortality	Total		10946	1368	12.5			
		Hypoxia							
			No	9244	678	7.3	1.00		
			Yes	1702	690	40.5	2.57	2.16	3.06
		Hypotension							
			No	9351	807	8.6	1.00		
			Yes	1595	561	35.2	2.89	2.54	3.29
	Disability	Total		10679	1707	16.0			
		Hypoxia							
			No	9058	912	10.1	1.00		
			Yes	1621	795	49.0	2.28	1.93	2.69
		Hypotension							
			No	9147	1094	12.0	1.00		
			Yes	1532	613	40.0	2.15	1.86	2.49

AOR, adjusted odds ratio; 95%CI, 95% confidence interval
* Adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, prehospital airway management, and new injury severity score.

Table 6 Interaction analysis of hypoxia and hypotension on mortality and disability using multivariable logistic regression analysis for sensitivity analysis

Group	Outcomes	Hypoxia	Normotension			Hypotension			P value for interaction
			AOR	95CI		AOR	95CI		
Study population excluding patients with SpO2<60% or SBP<60 mmHg									
	Mortality								
		No	1.00			1.00			
		Yes	1.25	1.15	1.36	1.38	1.19	1.62	0.0148
	Disability								
		No	1.00			1.00			
		Yes	1.29	1.18	1.41	1.40	1.19	1.65	0.0617
Study population using a new definition of hypoxia (<90%)									
	Mortality								
		No	1.00			1.00			
		Yes	1.90	1.74	2.06	3.16	2.75	3.62	<0.0001
	Disability								
		No	1.00			1.00			
		Yes	1.93	1.76	2.12	2.81	2.39	3.29	<0.0001
Study population excluding patients with traumatic brain injury									
	Mortality								
		No	1.00			1.00			
		Yes	1.78	1.60	1.97	2.44	2.05	2.90	<0.0001
	Disability								
		No	1.00			1.00			
		Yes	1.65	1.49	1.83	2.09	1.76	2.49	<0.0001

AOR, adjusted odds ratio; 95% CI, 95% confidence interval
Multivariable logistic regression analysis, adjusted for adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, pr ehospital airway management, new injury severity score, and interaction term(hypoxia*hypotension)

Also we tested the interactive association between hypoxia and hypotension for outcomes according to the level of the ED where the patients were transported. The EDs are classified into 3 levels; the level 1 EDs stand for Regional emergency centers, the level 2 EDs for Local emergency centers, and the level 3 EDs for Local emergency departments. In patients who were transported to the level 3 EDs, the AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on in-hospital mortality were 2.00 (1.65-2.44) and 3.48(2.49-4.87),

respectively. (p for interaction <0.0001) The AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on worsened disability were 1.88 (1.56-2.26) and 2.80 (2.00-3.90). (p for interaction <0.0001) In level 2 EDs, the AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on in-hospital mortality were 1.69 (1.51-1.89) and 2.35 (1.92-2.88), respectively. (p for interaction <0.0001) The AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on worsened disability were 1.39 (1.22-1.57) and 1.68 (1.34-2.10). (p for interaction = 0.0020) And in the level 1 EDs, the AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on in-hospital mortality were 1.32 (1.11-1.57) and 1.66(1.22-2.26), respectively. (p for interaction =0.0066) The AORs (95%CIs) by hypoxia according to non-hypotensive and hypotensive statuses on worsened disability were 1.36 (1.10-1.67) and 1.66 (1.13-2.42). (p for interaction <0.0001) (Table 7.)

Table 7 Interaction analysis of hypoxia and hypotension on mortality and disability using multivariable logistic regression analysis according to ED level

Group	Outcomes	Hypoxia	Normotension			Hypotension			P value for interaction
			AOR	95CI		AOR	95CI		
Lv3 ED									
	Mortality								<0.0001
		No	1.00			1.00			
		Yes	2.00	1.65	2.44	3.48	2.49	4.87	
	Disability								<0.0001
		No	1.00			1.00			
		Yes	1.88	1.56	2.26	2.80	2.00	3.90	
Lv2 ED									
	Mortality								<0.0001
		No	1.00			1.00			
		Yes	1.69	1.51	1.89	2.35	1.92	2.88	
	Disability								0.0020
		No	1.00			1.00			
		Yes	1.39	1.22	1.57	1.68	1.34	2.10	
Lv1 ED									
	Mortality								0.0066
		No	1.00			1.00			
		Yes	1.32	1.11	1.57	1.66	1.22	2.26	
	Disability								0.0533
		No	1.00			1.00			
		Yes	1.36	1.10	1.67	1.66	1.13	2.42	

AOR, adjusted odds ratio; 95% CI, 95% confidence interval
Multivariable logistic regression analysis, adjusted for adjusted for age, gender, mechanism of injury, Charlson comorbidity index, response time interval, prehospital mental status, pr ehospital airway management, new injury severity score, and interaction term(hypoxia*hypotension)

4. DISCUSSION

This study found a significant association between hypoxia with a SpO2 of less than 94% and in-hospital mortality and worsened disability in critical trauma patients who were treated by EMS. In addition, hypotension added a significant interaction effect with hypoxia on outcomes.

The effects of hypoxia and hypotension on mortality and disability were similar to a previous study performed in Arizona, USA.[11] The combined effect of hypoxia and hypotension measured by EMS on in-hospital mortality was significant higher (43.9%) than hypoxia only (28.1%), hypotension only (20.7%), and non-hypoxia and non-hypotension (5.6%), respectively. The adjusted OR in the combined risk group was 6.1 compared with the neither risk group. The study included a population who had moderate to severe traumatic brain injury. Our study included patients with all anatomical injuries as well as traumatic brain injury. The effect of hypoxia in our study subjects was similar to the effect in the TBI patients. Our data showed that the number of patients who received brain surgeries was 899 (5.2%) which was a relatively small number than what was reported in the population of the previous study (4 or higher in AIS-head scores equated to 44.5% of the sample). The severe to critical NISS group included 4707 (27.1%) patients, which was much small number than that reported in the previous study

(59.1%).[11] In our study, we found that the hypoxia effect was significantly associated to mortality in all types of trauma as well as in TBI. Our study analyzed the association between hypoxia and worsened disability as well as in-hospital mortality. Worsened disability was defined as a 2-point difference between the time of injury GOS score and the GOS score at the time of discharge. Even though our study population had a small proportion of TBI patients, the hypoxia effect was greater than mortality in the full model and the effect size increased in hypotensive patients.

Hypoxia was defined as a SpO2 of less than 90% in the above study, [11] which was different from the parameters that we set for hypoxia in our study (SpO2<94%). There is no clear definition of the level of SpO2 that is cut-score for what defines a hypoxic state versus a non-hypoxic statein trauma. Previous studies on airway management and adverse events have used the definition of a SpO2 of less than 90%.[22, 23] However, we used 94% as a cut-off because the national EMS trauma protocol of Korea recommends that airway management procedures should be initiated in patients who have a SpO2 reading of less than 94%. To compare the effect size of the hypoxia with cut-score value 90% in the sensitivity analysis, we tested for the association between hypoxia and in-hospital mortality and worsened disability (Table 5). The AORs for mortality and disability were much higher for hypoxic patients with SpO2 that were less than 94%. Our study discovered an association between the log

odds of death / disability and the SpO2 values (Figure 2). The figure shows the linear trend line in the adjusted full model. SpO2 values of 95% or higher did not show any increase in mortality or disability.

The previous study enrolled patients who had extremely low SpO2 values or SBPs.[11] Current noninvasive transcutaneous monitoring technology can measure extraordinarily low values of SpO2 or SBP. However, if a patient is profoundly hypotensive, SpO2 reading can be unreliable because of hypoperfusion in the peripheral tissues that can interfere with blood flow. Shock status can decrease the peripheral blood flow though vasoconstriction, which shunts blood to the vital organs.[24]During shock or resuscitation, the SpO2 may not be reliable. Because of this, profoundly low SpO2 and SBP values may be neither valid nor reliable. The previous study excluded patients with very low SpO2 (less than 10%) values or with very low SBPs(less than 40 mmHg). In our study, we enrolled all patients who had a recorded SpO2 (0-100%) and a recorded SBP (0-300 mmHg). To remove the measurement bias introduced by profoundly low SpO2 or SBP values, we performed a sensitivity analysis using the study population with both SpO2 readings that were higher than 60% and SBPs that were higher than 90 mmHg. Patients who were excluded by the new SpO2 and SBP criteria might be more severely injured or might have unreliable SpO2 or SBP values. From the sensitivity analysis, we confirmed these

results.

Detection of the SpO2 and SBP can by affected by the cold temperature, which can make the detection of the SpO2 and SBP difficult. To figure out the detection problem during winter season, we analysed excluded patients group due to unrecorded SpO2 or SBP. We categorized patients into three groups; Group 1(both values(SpO2 and SBP) are missing), Group 2(only 1 value missing), and Group 3(both values are recorded). Table 8 shows that there is no significant difference in seasons among three groups, which means the detection of the SpO2 and SBP at the scene is not affected by the cold weather.(Table 8.)

Table 8 Chi-square test to compare seasonal factor among the inclusion group and the excluded group with undetected prehospital saturation or blood pressure

Variables		All		both missing		1value missing		no missing		P-value
		N	%	N	%	N	%	N	%	
All		20111	100.0	1292	100.0	1413	100.0	17406	100.0	
season										0.8769
	Spring~Fall	14904	74.1	963	74.5	1041	73.7	12900	74.1	
	Winter	5207	25.9	329	25.5	372	26.3	4506	25.9	

Additionally, we tested the interactive association between hypoxia and hypotension for the subgroup population without any traumatic brain injury. The interaction was significant and effect size was significantly different for outcomes of non-TBI patients. These findings were consistent with those of whole population. Further study on preventing effect of prehospital hypoxia and hypotension on outcomes should be considered for patients with torso injury or extremity

trauma.

Also, the interaction was significant regardless of the ED levels. But higher level EDs showed relatively less effect size than lower level EDs, so we can suggest that the trauma patients with prehospital hypoxia and hypotension be trasported to the higher level ED for better outcomes.

The EMS system performance and protocol for trauma care is fundamentally important for outcomes. The EMS response time of these performances is critical for provision of optimal airway and supplement of oxygen for hypoxia patients. Our previous study showed the association between short prehospital time interval and worse clinical outcomes in severe trauma. [25] Our data showed the delay of response time interval was more in hypoxia than normoxia group. To reduce the hypoxic event, rapid response and optimal ventilation with oxygen will be the most important step in prehospital care for trauma.

We did not analyze the association between hyperventilation and outcomes. Hyperventilation cause the decrease the venous return and cardiac output. To avoid the hyperventilation, the end tidal CO2 level (ETCO2) should be measured. [26] The EMTs usually use the ETCO2 device when they put the ETI or SGA device in out-of-hospital cardiac arrest. In our study, however, only 0.2% patients received the advanced airway. Therefore, we could not analyze the effect of hyperventilation on outcomes in our study.

Fluid resuscitation is another crucial procedure in prehospital trauma care. [29, 30] However, we did not analyze the association between fluid resuscitation by EMS and outcomes. The intravenous fluid resuscitation was provided for the only 5.1% of study population (hypoxia 8.1% and hypotension 14.5%). There are many controversies on type of fluid, target value of blood pressure, and amount of fluid volume in prehospital fluid resuscitation. Korea EMS protocol for fluid resuscitation are preferred by level 1 EMT, rather than mandated for volume of fluid and type of fluid. [27]

Our study suggests that the hypoxia measured by SpO2 have much correlation with the outcomes in trauma patients, and the EMS protocol should focus to detect hypoxia and hypotension earlier and exactly for improving outcomes. In trauma patient, EMS protocols should contain the measurement of the SpO2, and protocols to detect the hypoxia and hypotension earlier should be emphasized. Education and training to prevent sustained hypoxic status through vigorous ventilation, oxygen supply within shortest time interval, and transport to the high level of EDs would be also critical.

Further study on impact of effective intervention or protocol change for hypoxia should be followed using a new technology such as high flow oxygen supply device or effective continuous positive airway pressure ventilation, which was tested on the efficiency in emergency department for non-traumatic respiratory problems. [28, 29]

5. Limitations

We selected a study population older than 15 years and excluded patients who received prehospital CPR, and who lacked SpO2 and SBP measurements. The criteria for our data selection could have impacted the final results.

The SpO2 and SBP measurements were performed by EMS providers (EMT level 1 or level 2), who can produce measurement bias through the use of different measurement devices, different methods, and different providers obtaining the measurements. Current devices for the transcutaneous monitoring of vital signs are manufactured by many companies. The devices should be permitted by the KoreanFood and Drug Agency (FDA) for the reliability and the validity of applied medical devices. All devices in this study were utilized as allowed by the Korean FDA.

The hospital care and outcomes were measured by medial record review. Even rigorous data quality management was required for the data registry, and the reviewers could utilize different measurements for the same condition, specifically GOS. We tested for reliability and validity during education sessions before the medical record review on the basis of simulation scenarios. However, we did not test the real data for reliability and validity. Our simulation tests did not produce measurement bias.

Finally, the study was performed in the EMS setting with an intermediate service level. The EMS protocol did not

encourage advanced care in the field or to remain on-scene in favor of initiating treatment (all scene times should be less than 10 min. except in cases of rescue operations). Because of protocol differences in this setting, the findings of the study may not be generalizable.

6. CONCLUSIONS

Hypoxia of less than 94% that is measured in the field was a significant risk factor for in-hospital mortality and worsened disability in severe trauma patients who were treated by EMS. An interaction effect with hypotension was also significantly different in a non-hypotensive patient group.

7. DISCLOSURE

The authors report no conflicts of interest

8. ACKNOWLEDGEMENTS

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요약(국문초록)

중증 외상 환자의 현장에서의 저산소증 및 저혈압이 병원 내 사망률과 장애 발생률에 미치는 영향 분석

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중증 외상은 대한민국에서 44세 이하 환자에서 사망 원인 1위를 차지하고 있다. 매년 30,000명 정도의 환자가 외상으로 인해서 사망하고 있으며, 많은 후유 장애를 남기게 된다. 중증 외상 환자에서 사망과 후유 장애를 줄이고 이를 조기에 평가하기 위해서 병원 전 단계인 사건 현장에서 구급대에 의해 측정 및 기록되는 변수들에 대한 분석이 필요함. 이중 현장에서 측정된 저산소증 여부가 사망률 및 후유 장애율에 영향을 미치는지에 대한 분석을 진행하였음. 또한 현장에서의 저혈압 여부에 따라서 이러한 저산소증의 영향이 증가하는지 함께 분석하였음.

2012년, 2013년 동안 구급대를 통해 전국 700여개 병원에 내원

한 성인 중증 외상환자 중 17,406명의 구급기록 및 의무기록을 수집하여 분석을 진행하였음. 구급대에 의해 측정된 저산소증 여부(산소포화도 94% 미만)를 독립변수로 설정하였고, 중증 외상환자의 병원 내 사망률 및 후유장애율을 결과 변수로서 설정하였다. 후유장애 발생 여부는 Glasgow Outcome scale의 2점 이상의 감소로 정의하였다. 교란요인 보정을 위해 다변량 회귀분석 및 interaction 모델을 사용하여 저산소증 및 저혈압 여부와 중증 외상환자의 사망률, 후유 장애율의 오즈비와 95% 신뢰구간을 계산하였다.

2012-2013년 중증외상환자 17,406명이 분석되었음. 이 중 14.9%에 해당하는 2,598 명이 사망하였고 21.5%에 해당하는 3,292명에서 후유 장애가 발생하였다. 또한 중증외상환자 중 16.7%에 해당하는 2,922명의 환자에서 사고현장에서 측정시 산소포화도 94% 미만의 저산소증이 확인되었음.

중증 외상환자 중 저산소증이 확인된 환자의 사망률(35.7%)이 그렇지 않은 환자의 사망률(10.7%) 보다 높게 확인되었고 후유 장애 발생 비율도 각각 51.2%와 15.9%로 저산소증이 확인된 환자에서 더 높게 확인되었다. 중증외상환자에서 저산소증 여부의 환자 사망 위험에 대한 오즈비는 2.15(1.92-2.40) 이었음. 또한 저산소증 여부의 후유 장애 위험에 대한 오즈비는 1.97(1.75-2.21) 로 확인되었다.

중증외상환자에서 수축기혈압 90mmHg 미만의 저혈압이 동반되었을 경우 저산소증 여부의 환자 사망 위험에 대한 오즈비는 2.66(2.32-3.04)이었다. 또한 저산소증 여부의 후유 장애 위험에 대한 오즈비는 2.17(1.87-2.53)로 확인되었다.

결론적으로, 중증 외상 환자에서 저산소증 여부가 환자의 사망 위험 또는 후유 장애 위험을 높이며, 저혈압이 동반된 경우 그 위험도가 더욱 증가하므로, 구급단계에서 산소포화도 및 혈압 등의 생체징후를 확인하여 환자의 중증도를 평가하고 적합한 치료를 받을 수 있도록 하여야 함.

주요어 : 중증 외상, 저산소증, 저혈압, 사망률, 장애 발생률
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